ART. VI.—*On Cold as a Cause of Acute Inflammation.* By A. F. A. KING, M.D., of Washington, D. C., Prof. of Obstetrics and Diseases of Women in the National Medical College, and in the University of Vermont; one of the attending physicians to Providence Hospital, Washington, D. C.

WHEN, after being chilled by exposure to cold, an individual suffers an immediate attack of acute congestion or inflammation (no matter of what organ), it is easy to infer, as is commonly done, that the "taking cold" produced the inflammatory attack. Thus, it is universally admitted that "*cold is an exciting cause of acute inflammation.*" In part, this is true; but it is only half the truth. Of two persons similarly exposed, one shall suffer nothing. There must therefore be present in the individual who is attacked some pre-existing condition, which entails upon him the *liability* to inflammation after exposure. And, further, since of several persons who thus suffer from acute inflammation, different *organs* may be attacked in each, it is evident there exists in the organs inflamed some pre-existing condition creating the liability to inflammation, and which does not exist in the organs that escape. Thus we are at once brought to acknowledge that cold, *per se*, is *not* a cause of acute inflammation; and it therefore becomes of great importance to ascertain what are the pre-existing conditions that create the liability to inflammation on exposure, and, in the absence of which, the inflammatory attack would not have occurred.

To define these (some of them) is the purpose of the present paper. And first let it be premised that I do not intend here to deal with such intense degrees of cold as produce frost-bite and the fatal torpor experienced by Arctic voyagers, etc. It ought, further, to be noted, also, that the frequency with which inflammation occurs is not so much in accord with a great *degree* of cold, as it is with *sudden depressions* of temperature, such as occur quite frequently during our winter season, or with sudden cooling of the body, during warmer seasons, from exposure to rain, and draughts of air, or from change of clothing, etc.

Of the bodily conditions that predispose to inflammatory attacks under such circumstances, we may mention:

First. Rapid Physiological Evolution. Children and young animals, though in quite a normal state, are peculiarly liable to congestion and inflammation on being exposed to cold, and hence with scrupulous care nature surrounds the young and growing progeny of all wild animals with the conditions that promote warmth; and the mother, in the case of the human species, takes the same precaution with her infant. That the condition of developmental growth is here the potent factor in predisposing to inflammation after exposure, is proved by the fact that the organs whose rate of development is at the time most rapid have the predisposition most marked. Thus, the child is more liable to inflammatory affections of the digestive and respiratory systems, than it is to those of the reproductive organs. On the other hand, later in life, as at puberty, the liability to inflammation of the reproductive system, on exposure, becomes very manifest. The buds of plants that have not yet entered upon their vernal growth withstand the frost, but those that, owing perhaps to a warm southern exposure, have begun to develop earlier, are killed by the first cold night. Eggs that have not begun embryonic development can withstand a degree of cold that would be fatal to the embryo after the developmental process has been set in action by the warmth of incubation. The increased mortality among young animals and infants during unusually cold seasons, especially if the winter has been prolonged to the breeding season, and accompanied with sudden vascillations of temperature, is well known. And there is no other way of accounting for this augmented mortality than that congestion and inflammation of some of the internal organs has occurred, and which, in the human species at least, we know to be the case.

Second. Inflammation following cold is peculiarly liable to occur in organisms that are undergoing *pathological evolution*, and especially in the particular organs which are the seat of more decided or rapid pathological development.

Before presenting evidence in support of this proposition, it is necessary to define what I mean by the term "*patholojical evolution.*" It will at once be surmised that the expression is meant to imply something analogous with *physiological* evolution. Such exactly is my meaning. As the normal development of a young organism is designed to secure the formation of certain structures, or organs, whose function, both in kind and degree, shall be such as to maintain a perfect adaptation between the organism and its natural environing conditions; so there also occur in the body other structural changes designed to secure a conservative modification both of structure and function, so that the organs thus modified may be adapted to other and *un*natural environing conditions to which the body has been subjected. These changes of structure I call processes of "pathological evolution." The causes that lead to them are the changes of environment just referred to. They are analogous with physiological developmental processes not only in the particular just mentioned, of securing adaptation, but also in being *gradual* in their formation ; *latent* as regards symptoms ; subservient in their growth to a given *type ;* and in being disastrously interfered with in their evolution by the same disturbing causes that interrupt physiological growth, and prominent among which latter must be mentioned *exposure of the body to sudden depressions of temperature*, as I may now proceed to demonstrate.

With persons in whom some chronic structural disease is *known* to exist, such as of the lungs, kidneys, stomach, etc., it is a common enough event for them to suffer acute inflammation of the affected organ, and to be finally "cut off" by such an attack, after exposure to cold. We constantly caution such persons to wrap up warm, and "take care of themselves," well knowing that if chilled, they are liable to congestion and inflammation of the chronically diseased organ. The frequency with which acute catarrhs and pneumonias thus terminate old cases of pulmonary disease is familiar enough; and so with other diseases. Chronic "invalids," for so we are wont to call such individuals, exhibit the same instinctive dread of cold as is observed in children and young animals, and the same liability to be easily chilled.

I have now to add that *unknown* structural diseases, viz: *latent, typical, gradual, pathological, evolutionary* changes, entail the same liability to inflammation on exposure, and in the absence of which (if we except other predisposing causes of inflammation presently to be mentioned), the inflammatory action would not occur. If this be true, it is of vast importance, inasmuch as the heretofore acknowledged frequent occurrence of acute inflammation in a previously perfectly healthy organ would be proved to be an error of no small magnitude. Furthermore, in the absence of the more usual evidence of chronic structural disease, the occurrence of inflammation would itself constitute a partial proof that such latent structural disease did really exist. There can be no doubt that many of the structural changes discovered after death, in organs that have just previously been acutely inflamed, are not at all due to the acute inflammation, but on the contrary preceded it and constituted the local condition predisposing to the inflammatory attack; this, too, notwithstanding the patient may have enjoyed good health, and have been free from any unpleasant symptoms directing attention to the altered organ. All this is easy enough of explanation when we recognize the fact that such designedly conservative processes of "pathological evolution," when pursuing their typical course undisturbed, are often completely *latent*. In no set of organs, perhaps, has the mistake of attributing post-mortem appearances to inflammation that ought not to be so attributed, been more prevalent than in those of respiration; and our entire pathology of phthisis and its kindred affections has been warped from the truth by this unfortunate error.

It is now very well known, however, since the researches of Handfield Jones, and others, that thickening and adhesions of the pleura, connective-tissue—hyperplasia, and consequent induration of the lung, and fibrous growths round the walls of the bronchi (peri-bronchitis), all of which, when found after death following acute inflammation, were thought at one time to be products of inflammation, are not always to be so accounted for, but very frequently occur slowly and latently without being suspected either by the patient or physician, and quite independent of inflammatory action.

True, it is frequently alleged that a patient, whose lungs were examined yesterday and presented no physical sign of latent or other disease, will to-morrow, perhaps, on being exposed to cold, suffer from acute pneumonia or pleurisy. Thus, it is believed, the inflammation attacked a perfectly healthy organ. From this I demur. If the individual had been perfectly well before exposure, why should he afterward be ill, while his companions escape; and if his lung had been previously sound, why should it now be inflamed, while other organs in the body have escaped inflammation? Evidently there was something wrong about the organ before the patient was exposed.

To say that physical examination revealed no signs of structural change before exposure is not sufficient. *It is impossible by any yet known means of physical examination to detect the beginning of disease in the thoracic organs; more likely it is only the very prodigious structural changes that are thus recognized.* To illustrate, let us suppose a case of chronic interstitial pneumonia, of incipient "fibroid phthisis," so-called, or "cirrhosis," a disease of very common occurrence, though formerly not so considered. This is acknowledged to be a very insidious affection, gradual in its development, and often quite latent. It consists chiefly in obliteration of the minute capillary vessels covering the respiratory surface of the lung, and in their substitution by hyperplasied connective tissue; in time all the normal structures of the affected part are supplanted in a greater or less degree by the same sort of fibroid growth. Can we detect the incipient or secondary stages of the affection, or, indeed, any other than what are in reality its later stages? I think not. Excluding adventitious sounds, râles, which would hardly be present in the beginning, certainly not in the absence of catarrh, there remain chiefly changes in the respiratory murmur and in the percussion resonance from which we should have to judge as to the existence or non-existence of disease. Experts in auscultation and percussion do not usually recognize more than three or four well-defined shades or degrees of modification in the normal resonance and normal murmur, although, practically, we all appreciate a certain number of intermediate or sub-grades of modification. Thus the percussion resonance is said to be "clear," "dull," or "flat," and the respiratory murmur "increased," "normal," "feeble," or "absent." If we could reduce the different grades of deviation in the resonance and respiratory murmur to an exact scale, it is questionable whether ever so acute and well-educated an ear could appreciate more than ten well-defined degrees of modification between health and the most exaggerated physical abnormity. That is to say, if the lung sounds in one individual were just normal, and those of another abnormal, in some degree less than one-tenth the abnormity in the latter case would scarcely be recognized with any certainty.

Returning now to our case of chronic interstitial pneumonia, let us suppose it has progressed to a sufficient degree to have obliterated and substituted by proliferated connective tissue *one square foot* of the rete of capillaries spread out over the respiratory surface; but this not in one spot, but diffused with tolerable equality over one lung. To have accomplished this thousands of capillaries and hundreds of air-vesicles must have been obliterated. Yet we cannot suppose that any so limited a change would be appreciable to the ear, for since, as the physiologist tells us, the normal respiratory surface in a full grown lung, if spread out, would cover seven hundred square feet (1400 *for both* lungs) we should have to recognize that the normal sound had been modified in a *one-seven-hundredth degree.* Even if the one square foot of obliterated surface were confined to one-seventh of the entire lung (the apex), instead of being diffused through the whole organ, we should still have to appreciate that the sound at the apex had swerved from the normal standard only in a *one-hundredth* degree. If, instead of one square foot, the extent of surface had been reduced by disease *ten square feet,* provided the abnormity were moderately diffused, it would still demand that we should recognize a departure from the normal sounds in a *one-seventieth* degree before the structural change could be diagnosed, yet here millions of capillaries and thousands of air-vesicles have been obliterated.

Nay, even if *fifty square feet* of the respiratory surface had been supplanted by connective tissue, provided the change were diffused, it is questionable whether the ear of every auscultator could recognize this prodigious abnormity, for it would require the recognition of a deviation from the normal sound only in a *one-fourteenth* degree.

Furthermore, when we reflect that the chest-walls in different localities and in different subjects vary from half an inch to an inch and a half or more in thickness, and that there exist in individuals that we acknowledge to be in perfect health certain variations in the size and shape of the thoracic cavity, and certain individual peculiarities in the size and functional capacity of the thoracic organs, it may justly be questioned whether these variations would not elicit more decided difference in the physical signs than could be produced by such limited organic changes as those just referred to as belonging to incipient chronic pneumonitis.

Of course, the same remarks apply with equal force to all other slowly-developing thoracic diseases.

Again, admitting a patient, whose lungs on recent physical examination have been pronounced healthy, to die shortly afterward with an acute pneumonia, and allowing that, on post-mortem examination, no evidence of previous structural disease of older date can be discovered, *there yet remains the doubt whether such previous structural change did not really* *exist;* for, aside from its being obscured by the products of acute inflammation, and even allowing the possibility of eliminating this source of obscurity, it is still questionable whether the limited modifications of structure, common to latent chronic pneumonia in its incipient and secondary stages, could be recognized by the ordinary *ma*croscopical method of conducting necropsies ; for it would require that modifications in the physical properties of the lung—in its colour, weight, density, elasticity, friability, etc. etc., from which examiners usually judge of abnormities—should be recognized when present only in a *one-seven-hundredth, one-hundredth, one-seventieth,* or *one-fourteenth* degree, respectively, for the several grades of pathological modification just mentioned when speaking of physical signs. Without *mi*croscopical examination they would most likely escape detection.

For the present, therefore, it certainly remains insufficiently proved that acute inflammation, after exposure to cold, can occur without the inflamed organ having been previously the seat of structural disease, or rapid growth, except under certain circumstances that constitute additional predisposing causes of inflammation on exposure now to be mentioned.

Third. Organs, previously quite healthy, but which have been subjected to violent, or too greatly prolonged functional exercise, especially if unaccustomed to such functional exertion, are liable to congestion and acute inflammation when the individual is soon afterwards exposed to cold.

This proposition is too evident to require much argument in its support. Exaggerated functional use is itself often sufficient to produce inflammation, but the addition of exposure to cold greatly enhances the liability to the acute attack, or increases its severity when it would have occurred without exposure. Acute inflammations having this origin must be familiar to every practitioner. I would only here add that the exalted nutrition consequent upon exalted function is in many particulars analogous with that belonging to physiological and pathological evolution.

Fourth. Organs are more than usually liable to acute inflammation on exposure to cold, *when they have been subjected to mechanical or chemical injury.*

And here, again, are going on processes of repair, in some sense analogous with physiological development, pathological evolution, and the exalted nutrition that follows exaltation of function.

In conclusion, it must be remarked that two or more of the above-mentioned predisposing conditions may be combined, when, of course, the liability to inflammation will be increased. Moreover, the existence of one of them creates a diminished toleration of the rest. Organs, for instance, undergoing physiological development, or pathological evolution, are thereby rendered less tolerant of exaggerated functional exercise, and of chemical or mechanical injury, and *vice versâ.*

From what has now been said, and remembering that certain individuals withstand almost any amount of exposure without injury, it may, I think, fairly be admitted that cold, *per se*, does not produce acute inflammation.

ART. VII.—*Three Cases of Penetrating Gunshot Wound of the Thorax, with Perforation of Lungs; Recovery.* By B. J. D. IRWIN, M.D., Surgeon and Bvt. Colonel, U. S. Army.

DURING the early part of the war waged between the United States and the *soi-disant* Confederate States of America, much valuable material pertaining to the surgical and medical history of the contending armies was rendered unavailable or utterly lost to science through various adverse circumstances that surrounded the hastily improvised armies of both sections. Prominent among the causes that interfered with the keeping and preservation of perfect records of all cases of injury and disease was the pernicious system that permitted vast numbers of invalid officers and soldiers to straggle away, with or without authority, from under the control of the medical department of the army, to receive professional care outside of the established military hospitals. If the histories of the sick and wounded of the armies of the North and especially those of the South who were never treated at their individual homes, hotels, boarding-houses, and other private places, could have been preserved, collated, and added to the statistical tables embraced in the "Medical and Surgical History of the War of the Rebellion," the inestimable value of this peerless work would be rendered still more precious by presenting deductions fortified by an increased array of illustrative examples.

Some of the information rendered inaccessible through the vicious cause adverted to may be recovered from time to time by individual efforts. As an evidence of this the following instances of severe gunshot injuries are submitted, the cases not having been heretofore reported in detail in the official returns made at the time, owing to the fact that the patients were treated at their homes or in private quarters.

CASE I. *Penetrating gunshot wound of thorax and abdomen, perforation of the lungs, diaphragm, and liver; recovery.*—Lieutenant F. P. James, Third U. S. cavalry, aged 23, of active habits and good constitution, while in charge of a party of skirmishers October 11, 1863, at Collinsville, Tennessee, was wounded in the following manner: Lying in the prone position he cautiously raised his head and chest to enable him to peer over the crest of the ground in his front, and while so doing was wounded by a rifle ball. The missile struck the anterior aspect of the right side of the thorax at a point three inches outside of the sternum, passed inwards and downwards, in a line with the position of the body at the moment, between the fourth and fifth ribs, impinging on and shattering